

**In the United States Court of Federal Claims**  
**OFFICE OF SPECIAL MASTERS**

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EUGENE MURRAY,

Petitioner,

v.

SECRETARY OF HEALTH  
AND HUMAN SERVICES,

Respondent.

\*\*\*\*\*

\* No. 19-1976V  
\* Special Master Christian J.  
\* Moran  
\*

\* Filed: October 6, 2022  
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\* entitlement, diagnosis,  
\* transverse myelitis,  
\* radiation myelopathy  
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Bridget McCullough, Muller Brazil, LLP, Dresher, PA, for petitioner;  
Zoe Wade, United States Dep't of Just., Washington, DC, for respondent.

**UNPUBLISHED DECISION DENYING COMPENSATION<sup>1</sup>**

Eugene Murray claims that the hepatitis B, haemophilus B, and/or tetanus diphtheria-acellular pertussis (“Tdap”) vaccinations that he received on June 14, 2017, caused him to develop transverse myelitis (“TM”). The parties have submitted reports from experts and argued their positions through legal briefs. Mr. Murray has not provided preponderant evidence showing that he suffered from TM. Further, the Secretary has provided compelling evidence that Mr. Murray more likely than not suffered from radiation myelopathy as a result of radiation treatment for his multiple myeloma (“MM”). Because Mr. Murray has failed to

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<sup>1</sup> The E-Government Act, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services), requires that the Court post this decision on its website. This posting will make the decision available to anyone with the internet. Pursuant to Vaccine Rule 18(b), the parties have 14 days to file a motion proposing redaction of medical information or other information described in 42 U.S.C. § 300aa-12(d)(4). Any redactions ordered by the special master will appear in the document posted on the website.

show that he suffered TM, he is not entitled to compensation. Thus, his case is dismissed.

## **I. Facts**

At the time he received the vaccinations at issue, Mr. Murray was a 66-year-old man with a relevant medical history of hypertension, hyperlipidemia, hyperthyroidism, diabetes mellitus, bilateral knee surgery, right rotator cuff repair, fusion of his L3-S1 discs, and deep vein thrombosis. Exhibit 1 at 4-7.

Mr. Murray's medical history also documented a diagnosis of multiple myeloma<sup>2</sup> in September of 2015 when it was found after a CT scan to assess Mr. Murray's lower back pain. Exhibit 2 at 37. After discovery of his myeloma, Mr. Murray immediately began radiation and chemotherapy. Id. at 92-95. Some of the radiation affected the T10 level. Id. at 39, 466, 526.

On June 21, 2016, Mr. Murray received a bone marrow transplant from which he recovered well. Id. at 691. On December 30, 2016, he reported to oncologist Dr. Shah for a six-month follow up on his bone marrow transplant. Id. at 861-64. Mr. Murray reported that he was suffering from continued peripheral neuropathy but denied any new bone pain. Id. at 863. Although Mr. Murray's cancer was in remission, his lab work revealed some marginally elevated levels suggestive of a relapse. However, Dr. Shah did not believe that they were high enough to be clinically significant. Id. Mr. Murray was given a favorable prognosis and received influenza and Prevnar 13 vaccinations before leaving. Id. at 866.

Mr. Murray's post-transplant treatment was relatively routine for a man in his condition. He returned to his oncologist for several follow up appointments, and on a June 6, 2017 appointment, reported that he was experiencing increased neuropathy in his hands. Exhibit 2 at 1039. On June 12, Mr. Murray returned to his palliative care specialist, Dr. Skold, reporting "pain starting in groin muscles bilaterally, radiating to the low back, [giving] it a 4 on the 0-10 scale." Id. at 1049. Dr. Skold noted tenderness to palpation in Mr. Murray's mid-thoracic and lumbar spine and was referred to physical therapy. Id. at 1053.

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<sup>2</sup> "[A] tumor composed of cells of the type normally found in the bone marrow." Dorland's Illustrated Medical Dictionary 1219 (32d ed. 2012).

Mr. Murray received the vaccinations at issue in this case two days later on June 14, 2017. Id. at 1065-66; Exhibit 13.

During Mr. Murray's June 12, 2017 visit, Dr. Skold ordered an "MRI for the 18th" of June in response to Mr. Murray's "[r]eworsening back pain." Exhibit 2 at 1050. On June 18, Mr. Murray underwent an MRI of the thoracic and lumbar spine with and without contrast. Exhibit 11 at 5. The findings of the reporting radiologist, Dr. Thanawala, showed "interval development of a small area of enhancement in the thoracic cord with associated edema at the T10 level which is new since the prior examination [of the 2/3/2016 MRI]." Id. at 6. Dr. Thanawala suggested this was a "nonspecific finding and would be a usual finding for multiple myeloma." Id. at 7. Dr. Thanawala also noted that there was a "redemonstration of mild neural foraminal stenosis primarily at the T9-T10, T10-T11." Id. at 6.

On July 13, 2017, Mr. Murray reported to the Blood and Bone Marrow Transplant Group of Georgia that the pain in his hip area began three to four weeks prior. Exhibit 1 at 50-52. He also reported neuropathic pain in his legs, feet, and fingers. Id. The following day, Mr. Murray reported to Dr. Vick-Bope at Southwood Pain Management. Id. at 77. Mr. Murray reported increased sudden and constant back, hip, and anterior thigh pain. He also reported a prior episode of similar pain in the past. Id. at 78. Mr. Murray's exam revealed decreased range of motion, pain with extension and rotation of the lumbosacral spine, negative straight leg test, mildly positive FABER test on the left and negative on the right. Id. at 82-83. Mr. Murray's lower extremities had normal strength and sensation, but symmetrically diminished deep tendon reflexes in the knees and ankles. Id. Dr. Vick-Bope reviewed Mr. Murray's June 18, 2017 MRI which revealed an "abnormal enhancement in the spinal cord at the T10 level," and "mild neural foraminal stenosis primarily at T9-T10 T10-T11 and moderate stenosis at T11-T12 levels." Exhibit 2 at 1087. She ultimately diagnosed Mr. Murray with lumbar post laminectomy syndrome, spinal stenosis of the lumbar spine, and multiple myeloma. Id. at 1091. She suspected that "the majority of [Mr. Murray's] pain is referred from the lumbar spine," and noted that she did not see any evidence of multiple myelopathy on exam. Id.

Mr. Murray received second doses of the hepatitis B and Tdap vaccinations on August 7, 2017. Exhibit 1 at 4.

On August 14, 2017, Mr. Murray was admitted to the Acute Care Center with symptoms of fatigue, malaise, muscle pain, and weakness lasting the previous four days. Exhibit 1 at 132-33. He also complained of increased urinary

frequency and explained that he had fallen on his way to the toilet that morning. Id. at 134, 144. Mr. Murray was subsequently transferred to Piedmont Healthcare Hospital. Id. at 144, 224-25.

During his admission, Mr. Murray complained of lower extremity weakness, burning pain in his thighs and buttocks, progressive bilateral lower extremity weakness lasting a month, and bilateral lower extremity spasms over the past week. Exhibit 1 at 240-42. Mr. Murray's exam revealed intact sensation and strength in his upper extremities, with slightly diminished strength in the lower extremities. Id. Mr. Murray received an MRI which revealed some enhancement at the T8-T9 level, and although this was noted to possibly represent demyelination, the treating physicians did not believe that it had anything to do with Mr. Murray's reported lower extremity symptoms. Id. at 241. Mr. Murray received a neurosurgery consultation on August 17, 2017. The neurosurgery exam was largely unchanged from Mr. Murray's previous exam, and the physician's assistant assessed Mr. Murray with lumbar stenosis secondary to progressive degenerative disc disease with foraminal stenosis. Exhibit 7 at 12. Importantly, upon discharge, Mr. Murray's discharge summary specifically notes that at this time, there was "no evidence to suggest acute [TM]," and that the evidence that was found pointing to demyelination would not have caused the lower extremity symptoms. Exhibit 1 at 241. Mr. Murray was discharged from Piedmont Hospital on August 18, 2017. Id. at 240.

Mr. Murray continued to report tingling and numbness in his lower extremities at an August 21, 2017 visit to internalist Dr. Desir-Joseph. Exhibit 1 at 247-50. He also complained of neck pain, and a limited x-ray showed advanced cervical stenosis at the C4-C7 levels. Id. at 251-52. On August 25, 2017, Mr. Murray reported to Southwood Internal Medicine that he continued to experience severe pain and weakness in his legs. He was again diagnosed with spinal stenosis and given an additional diagnosis of central nervous system ("CNS") demyelinating disease. Id. at 284. That same day Mr. Murray returned to his oncologist, Dr. Shah, for a follow up on his six-month myeloma study. Dr. Shah noted that Mr. Murray's myeloma was in remission and put Mr. Murray's Revlimid prescription on hold. Id. at 303.

On September 18, 2017, Mr. Murray was seen by Dr. Skold in palliative care for his bilateral thigh and lower back pain. Exhibit 1 at 376-77. Dr. Skold believed that the "likely culprit" for Mr. Murray's pain was his spinal stenosis, and

noted that “[e]verything that was described seems to be related to the Spinal Stenosis and Cauda Equina (Horse’s tail)<sup>3</sup> issue.” Id. at 377, 387.

Mr. Murray received a steroid and anesthetic injection to the L3 level of his spine on October 2, 2017. He reported nearly total relief of his pain and weakness. Exhibit 1 at 433. On October 11, 2017, Mr. Murray followed up with his urologist, reporting that he was doing well. Id. at 458.

Mr. Murray was next seen by neurologist Dr. Cole-Martin on October 30, 2017 for an evaluation of his demyelinating disease. Exhibit 1 at 488-94. Mr. Murray reported that his tingling and pain began in June of that year. Id. at 490. His exam revealed normal muscle bulk and tone, normal strength besides his left hip and left foot which were 4/5, deep tendon reflexes 2+, and pinprick test at about the nipple area. Id. at 493. Dr. Cole-Martin ultimately diagnosed Mr. Murray with TM and chronic inflammatory demyelinating polyneuropathy. Id. at 494. While it does appear that Dr. Cole-Martin was aware that Mr. Murray had a history of MM, she did not seem to have knowledge or consider Mr. Murray’s history of radiation therapy when arriving at her TM diagnosis. Id. at 488-94. Dr. Cole-Martin declined to recommend any treatment at the time, and deferred to Mr. Murray’s palliative care, oncology, and primary care physicians to decide on an appropriate workup “given underlying cancer.” Id. at 494.

On November 20, 2017, Mr. Murray was seen at Southwood Oncology complaining of increased falls, weakness, neuropathic pain in his lower extremities, low back pain, and urinary incontinence. Exhibit 1 at 569-75. Mr. Murray received MRIs of his cervical and thoracic spine on November 22, 2017. Id. at 529-31. His cervical spine MRI showed no abnormality while the thoracic MRI demonstrated “a mildly expansile region of central cord signal hyperintensity . . . slightly decreased in extent from the prior exam . . . A focus of enhancement in the left aspect of the cord at the level of T9 is no longer present.” Id. at 530. Mr. Murray’s radiologist, Dr. Wandler, believed that the results showed “no more than mild spinal canal and foraminal stenosis at T9-T10 and T10-T11.” Id. Mr. Murray received five rounds of IV steroids from November 29, 2017 to December 3, 2017. Exhibit 2 at 1381-1433.

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<sup>3</sup> “The collection of spinal roots that descend from the lower part of the spinal cord and are located within the lumbar cistern of the caudal dural sac; their appearance resembles the tail of a horse.” Dorland’s Illustrated Medical Dictionary 308 (32d ed. 2012).

Mr. Murray returned to Dr. Cole-Martin on December 5, 2017, reporting no improvement in his symptoms following his steroid treatment. Exhibit 1 at 643-58. Dr. Cole-Martin described Mr. Murray's case as "complicated" and recommended IVIG therapy and referred Mr. Murray to the MS Center of Atlanta "for question of TM in setting of previous chemotherapy and bone marrow transplant for cancer." Id. at 648. A CSF study from December 12, 2017 showed that Mr. Murray had an elevated protein level but no signs of inflammation. Id. at 696, 710, 717; Exhibit 4 at 27-31.

Mr. Murray received five IVIG infusions from December 12, 2017 to December 18, 2017. Exhibit 2 at 1470-1504.

On January 2, 2018, Mr. Murray was examined by Dr. Lawrence Morris at the Bone & Marrow Transplant Group of Georgia. Id. at 792-93. Mr. Murray explained that he had been diagnosed with TM and that he had received high doses of steroids and IVIG with little improvement. Id. Dr. Morris did not make any recommendations but noted that Mr. Murray's multiple myeloma remained in complete remission. Id.

On January 8, 2018, Mr. Murray returned to Dr. Skold who noted that Mr. Murray carried a new TM diagnosis and that IVIG therapy did not help. Exhibit 1 at 759. Mr. Murray also exhibited severe hemiparesis in his left leg. Id.

Mr. Murray was next seen by Dr. Williams at the MS Center of Atlanta on February 5, 2018. Exhibit 3 at 5-8. Mr. Murray's medical history lists that he began suddenly falling in July of 2017 and experiencing urinary incontinence. Id. at 5. Dr. Williams indicated that Mr. Murray's presentation was not suggestive of multiple sclerosis and ordered a round of lab work and diagnostic testing. Id. at 6. Mr. Murray was seen on follow up with Dr. Williams on February 27, 2018. Id. at 3-4. Dr. Williams concurred that Mr. Murray was suffering from TM, explained that there was no evidence of demyelination at the brain or cervical spine regions, and noted a specific period where Mr. Murray received five concurrent vaccinations. Id.

Mr. Murray did not exhibit any improvement between February and August of 2018. He received IVIG infusions, in February 2018 without effect, and began using a wheelchair in May 2018. Exhibit 1 at 874-79, 904-08; Exhibit 12 at 7178-81, 7214-16, 7387, 7548, 7569.



Mr. Murray received additional MRIs of his brain, brain stem, and thoracic spine on September 1, 2018. Exhibit 12 at 7570-72. Dr. Thanawala, the reporting radiologist, noted that Mr. Murray's TM seemed to have resolved, with no areas of abnormal spinal cord enhancement, and significantly improved signal from the midthoracic cord. Id. Dr. Thanawala also noted a stable, albeit moderate, degenerative disc disease in Mr. Murray's lower thoracic and upper lumbar spine. Id. at 7571, 7574. Mr. Murray's condition has since remained stable.

## **II. Procedural History**

Mr. Murray alleged that he suffered from TM as a result of his hepatitis B, haemophilus B, and/or Tdap vaccinations administered on June 14, 2017. Pet., filed Dec. 30, 2019. After a series of medical record submissions between December 30, 2019 and March 23, 2020, see Exhibits 1-14, this case was activated on April 22, 2020. Notice of Assignment at 1.

Mr. Murray filed an affidavit regarding damages on May 18, 2020, and additional medical records on May 26, 2020. Exhibits 16 & 17. Mr. Murray next filed an expert report from Dr. Frederick Nahm on August 14, 2020. Exhibits 17-28.

Dr. Nahm received his bachelor's degree in philosophy and neuroscience from the University of Michigan in 1986. He received his master's degree and Ph.D in neuroscience from the University of California – San Diego in 1990 and 1994 and his medical degree from the University of Michigan Medical School in 1996. Dr. Nahm is the founder and managing partner of NeuroCare Health P.C. in Greenwich, CT and an intraoperative neuromonitoring physician for NuVasive Clinical Services. Exhibit 18. Dr. Nahm has previously worked as the director of Greenwich Hospital's stroke program, as the medical director for Myomo Inc., and as a clinical assistant professor at Yale School of Medicine. Id. at 1. Dr. Nahm has held fellowships in electrodiagnostic testing at Harvard Medical school, a fellowship in medical ethics at the Harvard T.H. Chan School of Public Health, and a research fellowship at the University of Oxford. Id. at 1-2. Dr. Nahm's CV did not list any publications or research projects. Id.

Dr. Nahm opines that there are a variety of mechanisms by which Mr. Murray's vaccinations can cause TM including epitope spreading, polyclonal activation of B lymphocytes, and molecular mimicry, where a "foreign agent . . . can trigger an immunological response leading to the production of self-directed antibodies . . . that lead to the underlying biological injury and ensuing symptoms."

Exhibit 17 at 9. However, Dr. Nahm fails to explain which specific mechanism was at work in Mr. Murray's case. Instead, Dr. Nahm cites two case studies of hepatitis B vaccinees who suffered from TM as a way to show that there is a general association between the vaccine and the injury. *Id.* at 9-12. In the first case, hepatitis B surface antigens were found in the patient's spinal fluid. In the second case, circulating immune complexes containing hepatitis B antigens were found during the acute phase of the patient's TM and disappeared after functional recovery. Dr. Nahm suggests that these studies "provide empirical evidence that the Hep B vaccine can trigger an autoimmune response leading to factors that may play a role in spinal cord inflammation as in TM." *Id.* at 10. Dr. Nahm suggests that TM is an immune-mediated process due to TM biopsies showing high levels of immune cells in the spinal cord. He argues that because there was no evidence that Mr. Murray suffered from any overlapping neurological disorders such as neuromyelitis optica or multiple sclerosis, "the only explanation then is that the petitioner's idiopathic TM was the result of the multiple vaccines which he received prior to the onset of symptoms." Exhibit 17 at 11-12. Importantly, Dr. Nahm never discusses Mr. Murray's history of radiation therapy, nor the effect it may have on Mr. Murray's development of spinal cord inflammation.

The Secretary filed his Rule 4(c) report recommending against compensation on October 28, 2020. Resp't's Rep., filed Oct. 28, 2020. In his report, the Secretary argues that Mr. Murray's symptoms are best explained by his lumbar stenosis and radiculopathy. *Id.* at 13. He notes that two days before his vaccinations, Mr. Murray presented with "antalgic gait, and complained of bilateral pain in his groin muscles, radiating to the low back." *Id.* at 2 (citing Exhibit 2 at 1053). The Secretary highlights Mr. Murray's MRI records, noting that they revealed moderate neural foraminal stenosis at L3-L4, L1-L2, and L5-S1 levels as well as a small area of enhancement in the thoracic cord at the T10 level which was interpreted as "a usual finding for multiple myeloma" by Dr. Vick-Bope. *Id.* at 14 (citing Exhibit 2 at 1087). The Secretary further explains that Dr. Vick-Bope did not find "any evidence of myelopathy" at that time. *Id.* (citing Exhibit 2 at 1091). Additionally, the Secretary notes that an August 2017 hospitalization revealed "increased T2 signal and enhancement in a non-expanded thoracic spinal cord at T8 and T9," which the radiologist believed "could represent a demyelinating process [but] appear[ed] limited [to] two levels," making TM less likely. *Id.* (citing Exhibit 7 at 12-13). Addressing Dr. Cole-Martin's subsequent TM diagnosis, the Secretary argues that "the evidence does not reliably support TM as a diagnosis," highlighting Mr. Murray's preserved strength, deep tendon reflexes, and unresponsiveness to the steroid and IVIG therapies that are routinely used to successfully treat TM. *Id.* at 15 (citing Exhibit 1 at 387, 433, 490, 493-



494, 644, 759). The Secretary closes his report noting that although Dr. Nahm proposes a number of possible mechanisms by which he believes Mr. Murray's vaccinations could cause TM, he fails to offer any substantive discussion specific to Mr. Murray's case. Resp't's Rep. at 16. The Secretary notes with respect to prong 2 of Althen, none of Mr. Murray's treating physicians associated his injury with his vaccinations and that his initial symptoms preceded his vaccinations. Id. Thus, the Secretary concludes, even if Mr. Murray suffered from TM, he has not provided a reliable medical theory or logical sequence of cause and effect linking the vaccinations he received to the injury he alleges. Id. at 16-17.

Following the Secretary's report, Mr. Murray filed a supplemental expert report from Dr. Nahm with articles on April 1, 2021. Exhibits 29-31. In his supplemental report, Dr. Nahm clarifies his opinion, offering a more specific discussion on Mr. Murray's TM diagnosis and the biological mechanism at play. With respect to diagnosis, Dr. Nahm opines that the symptoms listed prior to Mr. Murray's vaccination were orthopedic in nature, and not neurological. Exhibit 29 at 1. Dr. Nahm explains that these symptoms were "of an entirely different nature than those which are being attributed to [TM]." Id. at 2. Dr. Nahm explains that the only symptoms attributable to Mr. Murray's TM were his muscle weakness, urinary frequency, muscles spasticity, and tingling across the thighs. Id. (citing Exhibit 1 at 227, 247-66). Further, Dr. Nahm explains that Mr. Murray's longitudinally extensive lesion in the thoracic spine from T7-T11 suggest he suffered from TM. He argues that Mr. Murray's CSF study showed "albuminocytological dissociation consistent with [TM]" as well. Id. (citing Exhibit 4 at 27-31). Dr. Nahm cites a diagnostic algorithm from the Frohman and Wingerchuk article that suggests Mr. Murray suffered from postvaccination TM because he had no signs of compressive lesions, no infection, systemic inflammatory autoimmune disease or cancer, no signs of neuromyelitis optica or MS, with an antecedent vaccination. Id. at 3 (referencing Exhibit 27 (Elliot M. Frohman & Dean M. Wingerchuk, Transverse Myelitis, 361 N.E. J. Med. 564 (2010))). Importantly, Dr. Nahm still fails to consider the effect that Mr. Murray's prior radiation therapy could have on spinal inflammation.

Dr. Nahm then clarifies that he believes molecular mimicry is the mechanism, at work in Mr. Murray's case. He highlights the studies from his previous report, and notes that there are "known homologies between certain epitopes of Hep B surface antigens, myelin basic protein . . . and myelin oligodendrocyte glycoprotein." Exhibit 29 at 4. He maintains that these proteins "have been strongly implicated in myelin damage in certain diseases such as MS," but does not specify that they have been implicated in TM. Id. Dr. Nahm cites a

study showing that 60% of hepatitis B vaccinees had a “Hep B surface antigen / MOG double reactivity at 3 or 6 months post-vaccination, compared to none before vaccination,” as a way to suggest that “a cross-reactive immunological response may occur between certain hepatitis B surface antigens, and myelin proteins, leading to demyelinating injury to the spinal cord.” *Id.* (citing Exhibit 31 (Dimitrios-Petrou Bogdanos et al., A Study of Molecular Mimicry and Immunological Cross-Reactivity Between Hepatitis B Surface Antigen and Myelin Mimics, 12 Clinical & Developmental Immunology 217 (2005))).

The Secretary filed a responsive expert report from Dr. Michael Wilson on June 1, 2021. Exhibits A-A.9; B. Dr. Wilson received his bachelor’s degree from the University of Chicago in 1998, and his medical degree from the University of California San Francisco (“UCSF”) School of Medicine in 2007. He also holds a Master of Applied Science degree in clinical research from UCSF. He completed his internship in internal medicine at Massachusetts General Hospital in 2008, and his residency in neurology with Harvard Medical School’s Neurology Residency Program in 2011. Exhibit B. He has held teaching positions at the Boston University School of Medicine, and the UCSF School of Medicine. Dr. Wilson has written 62 different peer-reviewed articles on demyelinating diseases, multiple sclerosis, and general neurology. *Id.* at 26-32. Dr. Wilson has been on the cutting edge of multiple medical discoveries, including a novel paraneoplastic disease and the first description at the single cell level of the combined transcriptional and immune repertoires of B cells in cerebrospinal fluid and peripheral blood of MS patients. Exhibits A & B.

Dr. Wilson opines that Mr. Murray’s TM was not the result of molecular mimicry triggered by his vaccinations, but rather the result of delayed progressive radiation myelopathy (“DPRM”) caused by the radiation therapy Mr. Murray received for his MM. Dr. Wilson explains that DPRM can begin within six months of exposure to radiation, but typically occurs within 12 to 15 months. Exhibit A at 6. Dr. Wilson further notes that it is well documented that DPRM patients have isolated elevations of total protein in their CSF, while TM patients also have elevated inflammatory markers. DPRM patients are also known to experience edema in the spinal cord and cord expansion as well as “heterogeneous gadolinium enhancement” on MRI as seen in Mr. Murray’s case. *See id.* Dr. Wilson emphasizes that these MRI changes are often associated with inflammatory disorders and therefore, DPRM can easily be mistaken for TM. However, according to Dr. Wilson, factors that distinguish DPRM from TM include: radiation exposure to vertebrae, insidious progression, and a lack of inflammatory markers with isolated protein elevation. *Id.* Additionally, Dr. Wilson highlights

the TM diagnostic criteria from Mr. Murray's own expert materials that state that for a diagnosis of TM, the patient must show "progression to nadir . . . between 4 hours and 21 days after symptom onset." *Id.* at 5 (citing Exhibit 27 (Frohman & Wingerchuck)). Further, and most importantly, the diagnostic criteria explicitly requires treating physicians to exclude, among others, "postradiation" causes. *Id.* Because Mr. Murray's progression lasted well beyond 21 days, and because no postradiation causes can be excluded, Dr. Wilson concludes that it is more likely that Mr. Murray suffered from DPRM, and not TM.

On June 30, 2021, Mr. Murray stated he did not intend to file any additional expert reports. Pet'r's Status Report, filed June 30, 2021.

After moving this case into the briefing phase, the undersigned issued a tentative finding denying entitlement pursuant to Vaccine Rule 5. *See* Order, issued July 9, 2021. In that finding, the undersigned explained that, as the record stood, he was "likely to find that Mr. Murray did not establish, by preponderant evidence, that he suffers from TM," thus defeating his claim. *Id.* at 3. The undersigned explained that advocacy and additional evidence may bring this finding into question and provided Mr. Murray with the opportunity to submit additional evidence before submitting a final brief on entitlement. Mr. Murray filed updated medical records on January 5, 2022, and a brief in support of entitlement on January 24, 2022. Exhibit 32; Pet'r's Br., filed Jan. 24, 2022. The Secretary filed a response to Mr. Murray's brief on April 18, 2022. Resp't's Br., filed Apr. 18, 2022. Mr. Murray did not file a reply brief, leaving the Secretary's argument unrebutted. Now that the briefing phase of this case has concluded, it is ripe for a decision on entitlement.

### **III. Standards for Adjudication**

A petitioner is required to establish his case by a preponderance of the evidence. 42 U.S.C. § 300aa-13(1)(a). The preponderance of the evidence standard requires a "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010) (citations omitted). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991).

Distinguishing between "preponderant evidence" and "medical certainty" is important because a special master should not impose an evidentiary burden that is

too high. Andreu v. Sec’y of Health & Hum. Servs., 569 F.3d 1367, 1379-80 (Fed. Cir. 2009) (reversing a special master’s decision that petitioners were not entitled to compensation); see also Lampe v. Sec’y of Health & Hum. Servs., 219 F.3d 1357 (Fed. Cir. 2000); Hodges v. Sec’y of Health & Hum. Servs., 9 F.3d 958, 961 (Fed. Cir. 1993) (disagreeing with the dissenting judge’s contention that the special master confused preponderance of the evidence with medical certainty).

When pursuing an off-Table claim, the petitioner bears a burden “to show by preponderant evidence that the vaccination brought about [the vaccinee’s] injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.” Althen v. Sec’y of Health & Hum. Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005).

Furthermore, as a threshold matter, a petitioner must establish he suffers from the condition for which he seeks compensation. Broekelschen v. Sec’y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). When a petitioner fails to establish his diagnosis, there is no need for an analysis pursuant to Althen, 418 F.3d at 1278. See Lombardi v. Sec’y of Health & Hum. Servs., 656 F.3d 1343, 1353 (Fed. Cir. 2011).

#### **IV. Analysis**

In this case, Mr. Murray argues that he suffers from TM while the Secretary contends that Mr. Murray suffers from delayed progressive radiation myelopathy. According to the Secretary’s expert, these injuries are similar in presentation, with entirely distinct etiologies and pathogeneses. Because of these significant difference in etiologies, these injuries, while similar in presentation, remain entirely divorced from one another, and therefore, the undersigned must first resolve the threshold issue of diagnosis as established in Broekelschen and Lombardi. See Broekelschen, 618 F.3d at 1346; Lombardi, 656 F.3d at 1353.

Mr. Murray alleges that he suffers from TM. However, the Secretary contends that his presentation, medical imaging, and test results are inconsistent with TM and more accurately reflect a diagnosis of radiation myelopathy.

## **A. Diagnostic Criteria**

The diagnostic criteria for transverse myelitis contain several elements that went unaddressed in Mr. Murray's filings. Indeed, Mr. Murray's expert introduced a "diagnostic algorithm" in his supplemental expert report, but did not provide a specific diagnostic criteria for TM. See Exhibit 29 at 3 (citing Exhibit 27 (Frohmman & Wingerchuck)). However, a clear and easy to apply diagnostic criteria that incorporates several elements missing from the diagnostic algorithm is present in the same Frohmman and Wingerchuck study on which Dr. Nahm relies. Mr. Murray's own materials suggest that the diagnostic criteria for TM require:

[(1)] Bilateral (not necessarily symmetric) sensorimotor and autonomic spinal cord dysfunction; [(2)] Clearly defined sensory level; [(3)] Progression to nadir of clinical deficits between 4 hours and 21 days after symptom onset; [(4)] Demonstration of spinal cord inflammation: cerebrospinal fluid pleocytosis or elevated IgG index, or MRI revealing a gadolinium-enhancing cord lesion; [and (5)] Exclusion of compressive, postradiation, neoplastic, and vascular causes.

Exhibit 27 (Frohmman & Wingerchuck) at 565. Thus, it appears that there are several criteria that Dr. Nahm failed to consider in assessing Mr. Murray's diagnosis, most importantly, the progression to nadir of clinical deficits between 4 hours and 21 days, and the exclusion of postradiation causes of Mr. Murray's injury.

## **B. Mr. Murray's Presentation**

The symptoms that Mr. Murray experienced are consistent with TM and radiation myelopathy, and thus, those symptoms alone offer little help at distinguishing his specific injury. However, the key to the inquiry lies in (1) Mr. Murray's medical imaging, (2) CSF studies, and (3) the progression of his injury to nadir, all of which are much more consistent with DPRM than they are with TM. In addition to reviewing these topics, the opinions of certain treating doctors are considered in paragraph (4).

### **1. Medical Imaging**

Because TM is caused by the inflammation of spinal cord sections, medical imaging of TM cases normally will reveal the necessary spinal cord inflammation. DPRM, on the other hand, is caused by white matter damage to the spinal cord and thus, inflammation is not always observed. Exhibit A at 6 (citing Exhibit A.1 at

101 (Timothy W. West et al., Acute Transverse Myelitis: Demyelinating, Inflammatory, and Infectious Myelopathies, 32 Seminars in Neurology 97 (2012)); Exhibit A.4 at 198-99 (P.J. Koehler et al., Delayed Radiation Myelopathy: Serial MR-Imaging and Pathology, 98 Clinical Neurology & Neurosurgery 197 (1996))). According to the Secretary's expert, it is "well documented that patients with radiation myelopathy can have not only edema in the spinal cord as evidenced by T2 hyperintensity, and cord expansion on MRI, but also heterogeneous gadolinium enhancement on post-contrast T1 MRI sequences." Exhibit A at 6 (citing Exhibit A.4 (P.J. Koehler et al.); Exhibit A.8 at 12-13 (Adams and Victor's Principles of Neurology (11th ed. 2019)); Exhibit A.9 at 1049 (Pao-Yu Wang et al., MR Imaging in Radiation Myelopathy, 13 Am. J. Neurocardiology 1049 (1992))).

Further, radiation to tissues adjacent to the spinal cord, including vertebrae, is a well-known cause of myelopathy, specifically referred to as radiation myelopathy. Exhibit A at 6 (citing Exhibit A.3 (R.J. Burns et al., Pathology of Radiation Myelopathy, 35 J. of Neurology, Neurosurgery, & Psychiatry 888 (1972)); Exhibit A.4 (P.J. Koehler et al.) at 199, 201; Exhibit A.5 at 247-48 (Shinobu Okada & Riki Okeda, Pathology of Radiation Myelopathy, 21 Neuropathology 247 (2001)); Exhibit A.6 at 118-19 (Jacques J. Palmer, Radiation Myelopathy, 95 Brain 109 (1972)); Exhibit A.7 at 415-16 (B. Sanyal et al., Radiation Myelopathy, 42 J. Neurology, Neurosurgery, & Psychiatry 413 (1979)); Exhibit A.8 at 12-13 (Adams and Victor's Principles of Neurology (11th ed. 2019)); Exhibit A.9 (Pao-Yu Wang et al.)).

A June 18, 2017 thoracic and lumbar MRI, performed shortly after manifestation of Mr. Murray's injury did not reveal any significant spinal cord inflammation, and the only abnormal enhancement was "nonspecific" at the T10 level and a "usual finding for multiple myeloma" patients. Exhibit 12 at 5052. Mr. Murray received radiation treatment at the T7 through T10 vertebrae, Exhibit 2 at 466, 526, the same vertebrae associated with his lower extremity symptoms, and the only vertebrae to exhibit any sort of abnormal enhancement on MRI. Ultimately, Mr. Murray's medical imaging reports offer little evidence in favor of a TM finding, as they do not exhibit much, if any, spinal cord inflammation that can be associated with TM. Conversely, the MRIs are much more consistent with what is to be expected of a patient who received radiation to their T7-T10 vertebrae. Consequently, the evidence preponderates in favor of finding that Mr. Murray's medical imaging results are more consistent with DPRM than they are with TM.



## 2. CSF Studies

Mr. Murray's CSF studies also suggest that he more likely than not suffered from DPRM than from TM. According to the materials submitted by the Secretary, it is "well documented that patients with radiation myelopathy have isolated elevations of total protein in the cerebrospinal fluid." Exhibit A at 6 (citing Exhibit A.1 at 107 (Timothy W. West et al.)). TM requires the presence of inflammatory markers. Exhibit 27 (Frohman & Wingerchuck) at 565.

Here, Mr. Murray had isolated elevated protein, without any elevated inflammatory markers. Exhibit 1 at 696, 710, 717; Exhibit 4 at 27-31. Dr. Wilson persuasively explained that isolated elevated protein levels are consistent with DPRM, while TM requires inflammatory markers. Further, Dr. Nahm failed to show that TM may present without inflammatory markers. As such, the evidence preponderates in favor of the Secretary on this issue, and that Mr. Murray's CSF studies are more consistent with a diagnosis of DPRM than with a diagnosis of TM.

## 3. Progression of Injury

Based on Mr. Murray's medical records, Mr. Murray's injury progressed for a significant period beyond what is defined by the TM diagnostic criteria. Without any additional argument from Mr. Murray on why or how TM may occasionally progress in the manner exhibited in Mr. Murray's case, the undersigned cannot find that the progression of Mr. Murray's injury is consistent with TM.

The diagnostic criteria for TM states that progression to the nadir of clinical deficits occurs within four hours to 21 days. Exhibit 27 (Frohman & Wingerchuck) at 565. Here, however, even construing the record in favor of Mr. Murray's argument, onset of his condition would have begun around June 21, 2017, when he began to experience increased urinary frequency, lower extremity weakness, and falls. Exhibit 1 at 132-34, 144. Mr. Murray reported frequent falls on November 20, 2017, and by January of 2018 he had withdrawn from physical therapy noting that his condition was "worsening, not improving." *Id.* at 569, 759. Thus, Mr. Murray's condition had yet to reach its nadir nearly six months after his initial vaccination, a period that is significantly outside that which is set by the TM diagnostic criteria. Mr. Murray's expert did not provide any explanation as to how or why this could happen in an abnormal case of TM, while the Secretary's expert persuasively explained that DPRM is a slow, insidious progression that can take months to develop. Exhibit A at 6.

#### 4. Opinion of treating physicians

Finally, it appears that the treating physicians who diagnosed Mr. Murray with TM were arriving at their diagnoses without ever considering Mr. Murray's history of cancer or radiation therapy. Although the opinion of treating physicians is given particularly favorable treatment in the Vaccine Injury Compensation Program, Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1326 (Fed. Cir. 2006), there is "nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it . . . cannot be rebutted." Snyder v. Sec'y of Health & Hum. Servs., 88 Fed. Cl. 706, 745 n.67 (2009).

As evidenced by the TM diagnostic criteria, TM is "a diagnosis of exclusion," requiring physicians to exclude specific causes of other neuropathic conditions, including post-radiation causes. See Exhibit A at 5; Exhibit 27 (Frohman & Wingerchuck) at 564; Exhibit A.2 at 500 (Transverse Myelitis Consortium Working Group, Proposed Diagnostic Criteria and Nosology of Acute Transverse Myelitis, 59 *Neurology* 499 (2002)). The two physicians who ultimately diagnosed Mr. Murray with TM, Dr. Cole-Martin and Dr. Williams, did so without making critical considerations.

Dr. Cole-Martin was the first physician to offer a diagnosis of TM. Exhibit 1 at 493-94, 496. However, her treatment records make no mention of Mr. Murray's history of radiation therapy, see Exhibit 1 at 488-92—a key factor in arriving at a TM diagnosis. Exhibit 27 (Frohman & Wingerchuck) at 565.

It appears that Dr. Williams also lacked Mr. Murray's MRI and CSF results, which would have excluded TM by revealing an increased protein level without inflammatory markers, and a spinal cord lesion inconsistent with TM. See Exhibit 1 at 865 (noting that Dr. Williams "[did]n't have medical records of [Mr. Murray's] MRI's. . . . [or his] LP . . . results . . . available for review."). Further, Dr. Williams was also seemingly unaware of petitioner's history of radiation therapy. Id. at 865-66.

According to Dr. Wilson, it would certainly be reasonable to diagnose a patient with TM if they had no history of radiation to their spine and showed signs of demyelination. However, TM must be excluded in favor of more consistent diagnoses when a patient shows no signs of spinal cord inflammation and inconsistent radiographic imaging such as what was exhibited in Mr. Murray's case. It appears, then, that all the treating physicians who diagnosed Mr. Murray

with TM did so without the medical history from Mr. Murray that would be most important in arriving at an accurate diagnosis. See Exhibit 1 at 488-92, 865-868. Consequently, the undersigned is unable to provide these records with the extra weight that is usually afforded to records of treating physicians. See Orloski v. Sec'y of Health & Hum. Servs., 147 Fed. Cl. 713, 725 (2020) (denying a motion for review and ruling that the special master did not abuse her discretion in not crediting an opinion from a treating doctor who did not address the diagnostic criteria), aff'd, 839 F. App'x 538 (Fed. Cir. 2021).

## **V. Conclusion**

Mr. Murray has not met his burden of demonstrating that he suffered from the condition, transverse myelitis, for which he seeks compensation. Accordingly, the Clerk's Office is instructed to enter judgment in accordance with this decision unless a motion for review is filed. Information about filing a motion for review, including the deadline, can be found in the Vaccine Rules, available through the Court's website.

**IT IS SO ORDERED.**

s/Christian J. Moran  
Christian J. Moran  
Special Master